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Radiotherapy, Radio-Diagnostics and Radiation Protection – How do They Differ?

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Differences between radiotherapy (RT), radio-diagnostics (RD) and radiation protection (RP) will be discussed with respect to their general aims, doses, dose-rates and volumes involved, and their dominant biological mechanisms. In RT the objective is to deliver a curative dose as high as reasonably achievable to a rather small target volume, in order to completely inactivate the metastasized cells within that volume, while maintaining the exposure of neighbouring critical organs or healthy tissues as low as possible (the "AHARA" principle). In RD and RP, conversely, the objectives are to obtain a clinically valid diagnostic image (RD), to realize exposures of radiation workers within their practices (RP), or to maintain the exposure of the general public (RP), with a dose as low as reasonably achievable (the ALARA principle). In particular, specified dose limits are not to be exceeded in RP, but not in medical exposures (RT or RD). Consideration of doses and exposed masses (or volumes, if density is accounted for) is important, as dose –the ratio of energy absorbed (in Joules) and mass of absorber (in kilogrammes) - being the ratio of two extensive (additive) quantities, is an intensive (generally not additive) quantity. In RT, exposing a target volume of , say, 1 kg to 60 Gy (typically in 30 week-day fractions of 2 Gy each) by penetrating radiation (e.g. gamma-rays) will inactivate all cells in the small, precisely irradiated, target volume. Within the context of RP, a high dose and dose-rate whole-body exposure (say, 60 kg) to 5 Gy of such penetrating radiation within minutes or hours, will fatally affect about 50% of the so exposed human population. An added complication in such exposures is the dose- and radiation quality-dependent relative biological effectiveness (RBE) of different types of radiation, such as protons or heavier ions. In all exposures, dose-rate is also relevant, since it is related to the time scales of biological mechanisms of inactivation and of rapid repair of radiation damage at the cellular and subcellular levels. Much slower are radiation-induced effects (such as repair) at higher systemic levels of the human organism, including recognition and possible elimination of radiation-mutated malignant cells by the immune system. The initial physical stages of interaction of ionizing radiation with human tissues take place within picoseconds, creating several radical species in the human cells. Here of particular interest are toxic reactive oxygen species (ROS) which may be created by background radiation, but are predominantly created as inescapable by-products of breathing oxygen by man. The rate of ROS production by respiration overwhelms that by natural background radiation by some 6 orders of magnitude. Since the human ROS-quenching systems is able to effectively handle the respiration-caused ROS, it will also effectively quench natural-radiation-induced ROS species – so long as the cellular repair capacities are not overwhelmed by high dose-rate exposures. The typical dose-rate in RT is 1 Gy/min., while that of natural background radiation – some 2 mGy/year, i.e. is lower by some 9 orders of magnitude. Under RT conditions, cells in the target volume will not be able to quench ROS production at that rate, so cells will die. At higher biological levels, cancer may also be initiated by ionizing radiation and result in cancers months or years later. In most cases, malignant cells may be recognised and destroyed by the immune system, but some will evade such detection. The general relationship between dose and radiation-induced cancer is certainly not linear, and the present linear-non-threshold (LNT) based system of radiation protection may require modification to incorporate recent advances in understanding radiation effects in man.

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